SCLEROSIS OF THE CORNU AMMONIS IN EPILEPSY.

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EPILEPSY is classed in text-books among functional nervous disorders. It is well known, however, that organic disease of the brain may give rise to convulsions bearing all the characteristics of epilepsy; and that sometimes such convulsions may be the only symptom of the disease.

In my service in the Arkansas State Lunatic Asylum and the Danvers Lunatic Hospital, I have examined the brains of forty-three epileptics. In only nineteen of these have I failed to find gross cerebral lesions. It is of one of these, sclerosis of the cornu ammonis or hippocampus major, that I wish to speak of on this occasion.

This condition was present, on one or both sides, in twenty of my cases. In eleven, no other abnormality was found; in nine, it was accompanied by other and more extensive lesions, which, in my judgment, had a common origin with it, and this association seemed to me to throw light on the nature of the connection between it and convulsions.

A number of observations of this kind may be found scattered through earlier medical literature, but the lesion was, I believe, first brought into prominence by Meynert, who published,[1] in 1868, brief reports of twenty such cases dissected by him. In regard to its connection with the symptoms, he expresses himself as follows:

"For the present, I will only remark that it is very unlikely that the primary cause of so common a disease is to be found in the equally common primary affection of so limited a region of the cortex as the cornu ammonis, while frequently enough, in addition, extensive, apparently far more essential and not so strictly localized, brain diseases are present in epileptics.

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[1] Vierteljahrsschrift f. Psychiatrie, Bd. I., S. 395.

"I hold the supposition, therefore, to be more warranted, that, through certain anatomical relations, the cornu ammonis becomes involved in disease in consequence of epilepsy produced by quite remote lesions."

Since Meynert's report was published, observations of this kind have been numerous, and I am not aware that any observer who has sought systematically for this lesion in epileptics has reported a failure to find it in a considerable proportion of cases. Sommer[2] publishes a table of ninety observations, only one of which is personal. In this case, the patient, a man aged twenty-five at death, had suffered for ten years from attacks of petit mal. Mentally, his condition was characterized by dementia with attacks of excitement. At the autopsy, the points of both temporal lobes were found softened, and the right cornu ammonis was of cartilaginous hardness. Microscopically, the author found atrophy of nervous elements, proliferation of connective tissue, obliteration and stenosis of blood-vessels, and a considerable reduction in number of the large cells of the fascia dentata. Adopting Ferrier's view, that the cornu ammonis is the centre of common sensibility for the opposite side of the body, he believes that disease of that portion of the brain excites paræsthesiæ, which, in predisposed persons, may act in the same way as peripheral irritations in producing convulsive attacks. He finds disturbances of sensation mentioned in thirty-eight of the cases in his table, and only explicitly denied in three.

Coulbault[3] has collected ninety-three cases, largely identical with Sommer's, five of which seem to have been previously unpublished. He gives the microscopical findings, by Bouchard, in one. The patient was a woman, aged 66, said to have been subject to "weaknesses" for twenty years; to epileptic attacks for six years. Daily attacks of petit mal; severe convulsions about once a month. Death from cholera. The right fascia dentata was of very hard consistency, and presented, microscopically, multiplication of nuclei in capillaries and arterial adventitia, numerous nuclei and corpora amylacea in the cerebral substance. The pyramidal cells were few in number and pigmented. Left fascia dentata healthy.

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[2] Arch. f. Psych., Bd. X., S. 631.

[3] Des Lesions de la Corne d'Ammon dans l'Epilepsie. Thèse de Paris, 1881

He formulates his conclusions as follows:

1. Lesions of one or both cornua ammonis occur in certain epileptics.

2. These lesions vary in character. The most common is sclerosis, with or without atrophy. Less frequently they consist in softening or in tumors of various nature.

3. Lesions of the cornu ammonis are frequent in epilepsy.

4. Observations show that these lesions are accompanied by various disturbances of general and special sensibility.

5. Lesions of the cornu ammonis are a cause, not a consequence, of epilepsy.

6. The histological character of the lesion seems to consist principally in hyperplasia of connective tissue, resulting in atrophy and disappearance of pyramidal cells.

Hemkes[4] found six examples of this lesion in autopsies on thirty-four epileptics. On the ground of results of experimental extirpation and irritation of the cornu ammonis, by himself and Nothnagel, he concludes that the sclerosis cannot be the cause of the convulsions, which he is inclined to attribute to a localized encephalitis, involving this convolution among other parts. He adduces the following facts in favor of this view: In all his cases of this kind the convulsions began at an early age. In five, there was a history of preceding disease—infantile convulsions in two, typhus, meningitis and scarlatina in one each. In all, the brain weights were much below the average, either for healthy persons or epileptics.

Féré[5] has met with this lesion twelve times. He thinks the known facts of cerebral localization hardly favorable to the view that it is the cause of the convulsions, although the association is too frequent to be considered a mere coincidence. He suggests that it may be a predominant localization of a more extensive sclerosis.

Chaslin[6] gives an account of the histological examination of the brains of four epileptics. One presented no lesion to the naked eye; in the others there was sclerosis of various convolutions, including the cornua ammonis, which latter, however, do not seem to have been examined microscopically. He found atrophy of the nervous elements, with overgrowth of neuroglia and development of spider cells. These changes existed in a minor degree in the brain, which appeared normal to the naked eye. The principal point which he makes is that the hypertrophied elements are not connective tissue, in the ordinary sense of the term, but are derived, like the nerve cells, from the ectoderm. He expresses no opinion as to the nature of the connection between the lesions and the symptoms.

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[4] Allgem. Zeitschr. f. Psych., Bd. XXIV., S. 678.

[5] Les Épilepsies et les Épileptiques, Paris, 1890, p. 441.

[6] Arch. de Méd. Expérim., May 1, 1891.

Fischer[7] found, in a man, epileptic from his seventh year, who died at the age of fifty, atrophy, moderate in degree, of the frontal and occipital lobes of both hemispheres, and of the left temporal lobe, with extreme atrophy of the left cornu ammonis. Prof. Hoffman, who made the histological examination, reports atrophy, in the latter convolution, of both white and gray substance; the different layers of the latter were equally affected. The medullated fibres entering the hilum of the fascia dentata were very few in comparison with the opposite side. The blood-vessels were healthy, and there was no appearance of a primary gliosis. He considers the lesion of the hippocampus a part of the general atrophy of the hemispheres, and is disposed to accept Wundt's explanation of the preponderance of this process in the cornu ammonis, according to which it is due to dilatation of the lateral ventricle, arising from disturbance of the circulation. At the same time, he admits that there was no conspicuous difference in the size of the ventricles in his case. He also reports, without histological examination, a case in which there was great asymmetry of the hemispheres, the right weighing 560, the left 685 grammes, with atrophy of the right cornu ammonis, and another in which, after six years' duration of epilepsy, with advanced dementia, the left cornu ammonis was found broader, flatter and softer than the right.

Bourneville and d'Olier[8] describe a case of induration of the end of the left cornu ammonis in a young woman who suffered from convulsions in infancy, ceasing at five years of age, and returning, this time permanently, at fifteen. They give no description of histological appearances, and express no opinion as to the relations of the lesion to the convulsions.

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[7] Festschrift zur Feier des fünfzigjährigen Jubiläums der Anstalt Illenau. Heidelberg, 1892. S. 127.

[8] Arch. de Neurologie, I., p. 213.

Pfleger,[9] in autopsies on forty-three epileptics, found atrophy and sclerosis of the cornu ammonis twenty-three times, atrophy without sclerosis twice. Of these cases seven were men and eighteen women. The right cornu was affected in sixteen cases (three men, thirteen women); the left in six (two men, four women), and both in three (two men, one woman). During his studies and his service as demonstrator of anatomy at the Vienna School, he had never found this lesion. Among about 300 autopsies in the poor-house (Versorgungsanstalt) at Ybbs, where these observations were made, he found it in two other cases—one, a male, of paretic dementia (bilateral atrophy and sclerosis), and a woman, aged 78, hysterical and subject to convulsions without demonstrable loss of consciousness.

He is disposed to attribute the lesion to a disturbance in the circulation of the convolution during and after the attack.

Nerander[10] gives an account, which I have seen only in abstract, of five cases of changes in the cornu ammonis in epileptics. Sclerosis seems to have been pronounced in only one of them. He found similar lesions in other parts of the brain of one, and in several, degenerative changes in the blood-vessels. He is disposed to think that sclerosis and atrophy in this region may be defects of development originating in fœtal life or early infancy. He thinks they have no special relation to epilepsy, as they are not infrequent in persons who are not epileptic. He found more or less pronounced changes in one or both cornua ammonis in eight out of fourteen epileptics in the Lund Hospital for Insane, and during the same time no less than twelve cases of alterations of the cornu ammonis in non-epileptics; has also found differences in size between the two cornua in non-epileptics.

The latest article of importance on the pathology of epilepsy which has come under my notice is that of Blocq and Marinesco.[11] These authors examined the medulla oblongata and the Rolandic convolutions in nine cases of

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[9] Allgem. Zeitschrift f. Psychiatrie, XXXVI., S. 359.

[10] Studien öfuer Förändriugarna i Ammonshornen och Närligganda delar vid Epilepsi, Lund, 1894.

[11] Semaine Médicale, Nov. 12, 1892, p. 445.

"essential" epilepsy dying from that disease. In the cerebrum they found in every case, in specimens stained according to Marchi's method, infiltration of the perivascular sheaths with granule cells, and granules, staining black, in the cells of the superficial layer of neuroglia. These changes they believe to be the result of phagocytosis of products of degeneration. By other methods of staining they failed, in four cases, to find anything pathological; in four, there was diffuse proliferation of neuroglia, of greater or less extent, with evidence of vascular disturbance, and in one, disseminated foci of sclerosis, with dilatation of blood-vessels and punctiform hemorrhages, both in the cerebral cortex and the medulla oblongata. With the exception of this last-mentioned case, the medulla was found healthy in all. They do not allude, specifically, to sclerosis of the cornu ammonis, but conclude that the lesions described by Chaslin, in the article already cited, are not, as the latter, with Féré and Marie, supposes, primary, but are the results of the attacks, as they believe to be the case with the changes which they themselves describe. They conclude that there is, in so-called essential epilepsy, an abnormal excitability of the motor centres of the cortex, of which the anatomical basis is as yet unknown; that the exciting cause of epileptic convulsions, in some cases at least, is an auto-intoxication, and that the convulsions may have anatomical changes for their result.

It appears from the foregoing, that the preponderance of authority is in favor of the view that the lesion in question is a result, rather than a cause, of the convulsions with which it is associated.

Before proceeding to the account of the individual cases of my series, it may be well to give a general account of the lesions common to them all, and which were found surprisingly uniform.

Macroscopically, in every case observed by me, the gyrus hippocampi (subiculum cornu ammonis), as well as the hippocampus itself, was reduced in size in all its dimensions—in length as well as thickness. The subiculum did not present any very marked change of consistency, but the cornu itself was always firmer than natural—sometimes of a consistency resembling cartilage.

In carmine-stained sections, no marked histological change appears in the gyrus hippocampi, either in the cells, fibres or neuroglia, until the deeper layers pass into the compact layer of large pyramidal cells known as the stratum pyramidale (stpy). In this layer there is, in every case, absence of most of the characteristic cells and great diminution of the area of the layer. In some cases, the cells are comparatively well preserved for a limited extent. In this case, it is always at the same point—just before the entrance of the stratum pyramidale into the nucleus of the fascia dentata (stpy, Fig. II).

The stratum pyramidale is continuous with the nucleus of the fascia dentata (nfd), and the latter contains, normally, great numbers of irregularly arranged, large, multipolar cells, similar, in general appearance, to those of the former layer. In all the cases of sclerosis, the nucleus is greatly diminished in size, and contained very few of the characteristic cells (nfd, Fig. II).

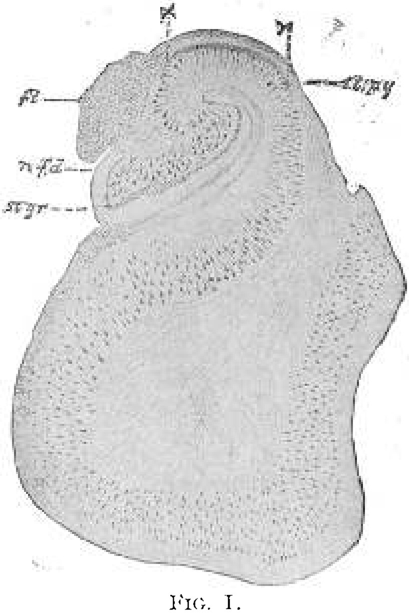

FIG. I.

The stratum pyramidale and nucleus fasciæ dentatæ in the diseased convolutions are occupied by a dense, finely fibrillated neuroglia, staining more deeply with carmine than the normal tissue. It contains numerous nuclei, but, in most of the cases, comparatively few "spider cells."

Surrounding the nucleus of the fascia dentata, except at the hilum, is the stratum granulosum (stgr), a thick layer of small pyramidal or bipolar cells, with apices directed outward. In most cases this was decidedly thinner than in normal specimens, and in some the layer was absent in a considerable part of its extent.

The changes in the white substance can be best studied in specimens stained after Weigert's or Pal's method. In such specimens the principal changes are found in the alveus (alv). This consists, as is well known, of two layers. The more superficial, thicker and denser, is composed of fibres mostly running lengthwise of the convolution, and, consequently, cut transversely in a cross section. This layer appears normal in structure, although somewhat thinner than in health, in the sclerosed convolutions. The deeper layer, composed of fibres cut longitudinally in cross section of the convolution, is, on the contrary, almost entirely wanting (see Figs. III. and IV., a). This layer is continuous at the hilum of the fascia dentata (hfd) with a large pencil of medullated fibres emerging from the nucleus. These fibres are also very few in number in the nucleus fasciæ dentatæ of the diseased convolutions (hfd, Fig. IV.).

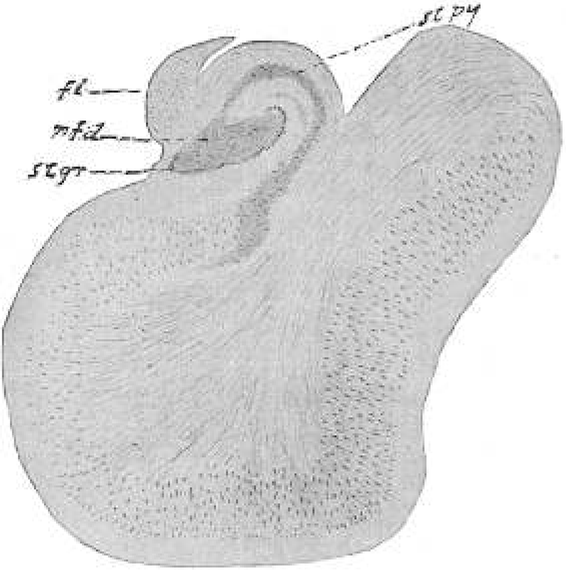

FIG. II.

The deeper layer of the alveus is contiguous to the bases of the large pyramidal cells of the stratum pyramidale. The simultaneous absence of these cells and the nerve-fibres of this layer justifies, it seems to me, the assumption that the latter are the axis-cylinder processes of the former, and that the large bundle of medullated fibres found normally in the hilum fasciæ dentatæ bears the same relation to the nerve-cells of the nucleus. It may, then, be said that the most conspicuous histological change in these cases is the disappearance of the large nerve-cells of the stratum pyramidale and nucleus fasciæ dentatæ, with the nerve fibres formed by their axis-cylinder processes.

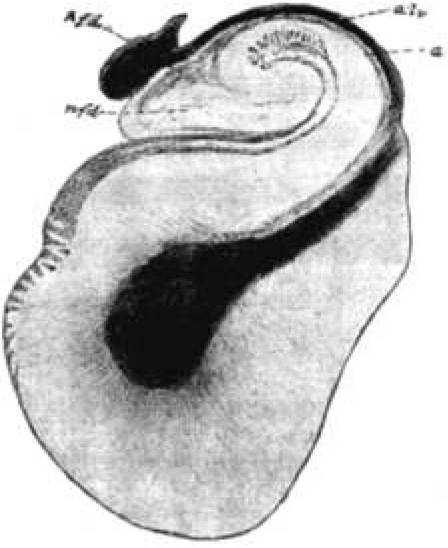

FIG. III.

Proceeding to the report of my own cases, I will first describe the cases in which the disease of the cornu ammonis was associated with other gross lesions.

Case I.—E. H., white, male, aged 19 at death, an inmate of the Arkansas asylum for seven years. This was a case of infantile hemiplegia with contractures, affecting the left extremities, which were somewhat smaller than the right and much limited in movement. There were athetoid movements of the fingers and toes. He had a depressed cicatrix of the scalp, a little to the right of the median line, deepest just anterior to the coronal suture, and extending backward for a short distance over the adjacent portion of the parietal bone, which he attributed to a blow from a falling rail when he was two years old. He was considerably demented and had frequent attacks of haut mal. Death in status epilepticus, July 11, 1891.

At the autopsy, on removal of the scalp, an opening, about half an inch in diameter, which had escaped observation during life, was found in the frontal bone, under the most depressed part of the cicatrix. It was circular in shape and bevelled at the expense of the outer table. There was no depression of surrounding bone, no appearance of splintering, and no abnormal adhesion of the dura mater either to the bone or the arachnoid. The cerebral hemispheres were healthy in appearance, with the exception of the right cornu ammonis, which was much smaller and harder than the left. The right corpus striatum, optic thalamus and cerebral peduncle were atrophied, sclerotic and of a yellowish color.

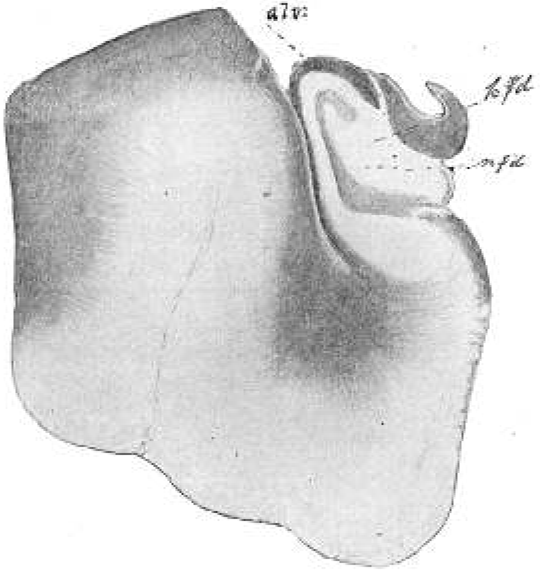

FIG. IV.

Microscopically, the part of the cerebrum lying beneath the opening in the skull presented no distinct evidence of disease. The left cornu ammonis seemed healthy, but in the right the pyramidal cells of the stratum pyramidale are wanting in its whole extent, except near the point of entrance into the nucleus, where a few are to be found. The nucleus itself is almost destitute of cells, and very much shrunken; the stratum granulosum much thinned and wanting in places. Nuclei are numerous in the neuroglia of the sclerosed portions, but spider cells wanting.

Case 2.—S. B., white, single woman, aged 46 at death, inmate of the Arkansas asylum for two and one-half years. Had talipes varus of right foot, and paralysis with contracture of both extremities of that side. She was almost completely idiotic; her vocabulary probably did not contain over twenty words, very imperfectly pronounced. Convulsions severe, but not very frequent. Death December 4, 1891, of pulmonary tuberculosis, with pyopneumothorax.

At the autopsy, extreme asymmetry of the two sides of the brain was found. The total weight of the brain, with the membranes, but without cerebro-spinal fluid, was 33 ounces. The right hemisphere, separated by Meynert's method, weighed fifteen ounces; the left, nine. The convolutions of both hemispheres were pretty regular in type, and there was no marked difference in consistency, except that the left cornu ammonis was harder in texture as well as smaller than the right. All the convolutions of the left hemisphere were small, with wide sulci. The basal ganglia of the left side were smaller than those of the right, about in proportion to the corresponding hemispheres, and the optic thalamus was of firmer consistency. Cerebellum, pons, medulla oblongata and spinal cord presented no very obvious anomaly to the naked eye.

Microscopically, the convolutions of the right side, inclusive of the cornu ammonis, show little variation from the normal condition, although there is a spot in the gyrus hippocampi which seems deficient in large pyramidal cells. In the left, the stratum pyramidale is interrupted in several places, and the deficiency in large pyramidal cells extends over the convexity of the gyrus hippocampi; the nucleus fasciæ dentatæ is almost destitute, and the stratum granulosum very deficient in their respective cellular elements. The character of the sclerosed tissue is much the same as in the previous case. In the remaining convolutions of the left hemisphere, sections from different parts fail to show any very conspicuous change in the structure of the gray matter of the cortex, which seems to be nearly or quite as thick as in the right hemisphere. The deficiency seemed to be mainly in the white matter, in which the nuclei are more abundant than in normal brain.

Case 3.—L. M., white, single woman, aged 31 at death; a resident of the Arkansas asylum for nine years. Another case of infantile hemiplegia, affecting the left side. Upper extremity contractured, ill-developed and almost useless; lower much smaller than right and very much limited in movement. She was subject to not very infrequent attacks of grand mal and to occasional seizures in which, without loss of consciousness, the extremities of the affected side would be, for hours, agitated by clonic spasms. These could be checked by a full dose of chloral hydrate. No history of the origin of her paralysis was furnished. Mentally, she was irritable and imbecile, but had no defect of speech, and was able to do a little work. Without being known to have had recent convulsions, she passed gradually into a comatose state and died January 10, 1892.

The autopsy revealed a very similar condition to that found in the previous case. The brain weighed 37 ounces; right hemisphere, separated by Meynert's method, nine ounces; left, seventeen ounces. The right hemisphere was everywhere of much firmer consistency than the left; the temporal lobe, especially, of almost cartilaginous hardness. The basal ganglia were smaller on the right side than on the left, in about the same proportion as the hemispheres.

Microscopically, in the right hippocampus, there is almost complete absence of nerve-cells in the fascia dentata, both in the nucleus and the stratum granulosum. The stratum pyramidale is interrupted at one point, near its origin, and is ill-developed throughout. In the hemisphere in general, the cortex does not seem much, if at all, thinned, but there is apparently some deficiency of nerve-cells, more especially in the fifth layer. Spider cells are abundant, particularly in the white matter. Nerve fibres are abundant in the cortex, as shown by Pal's staining, but the tangential fibres of the superficial layer are much finer than usual.

Case 4.—T. M., white, male, aged 14 at admission, resident of Arkansas asylum seven years. Case of infantile hemiplegia, with epilepsy; duration and origin not stated. Right extremities slightly smaller than left; arm and hand contractured; athethoid movements of fingers; spastic gait. Had an attack of dysentery, from which he seemed to recover, but failed in strength and died April 15, 1894.

At the autopsy the cranium was found to be asymmetrical, the left side being distinctly smaller than the right. The left hemisphere of cerebrum was smaller in all dimensions than the right; temporal lobe and insula distinctly firmer in consistency; cornu ammonis sclerotic, shrunken and hard. Separated by Meynert's method, right hemisphere weighed twenty ounces; left, fourteen and a half ounces. Weight of brain, forty-four and a half ounces. No very noticeable difference in corpora striata. Left optic thalamus not much more than half the size of right; harder in consistency. Cerebral substance, except as above mentioned, of normal consistency and color; blood-vessels healthy.

Case 5.—F. S., a woman, aged 46, died in the Danvers Lunatic Hospital, June 29, 1895, in status epilepticus. She was said to have had a "shock" at the age of five years, ever since which time she had been paralyzed in the right side and subject to convulsions.

The hemiplegia was of moderate severity, leaving her some use of the hand. She was imbecile, but could read. Convulsions not very frequent.

At the autopsy the cranium was asymmetrical, the left side being smaller. The anterior part of the falx cerebri was wanting, and the arachnoid of the frontal lobes was adherent on their adjacent surfaces. The left hemisphere was smaller in all its dimensions than the right, especially behind the fissure of Rolando. The posterior third of the corpus callosum was reduced to a translucent membrane. The left lateral ventricle was greatly dilated. In the parietal region, at the bottom of the sulci, the cerebrum was about one-eighth of an inch thick. The entire hemisphere was of firmer consistency than the left and of lower specific gravity, as shown by a tendency to float in the hardening fluid. The left cornu ammonis was not more than half the size of the right and very much harder. The left corpus striatum was decidedly smaller than the right, and the left optic thalamus not more than half the size of its fellow. The brain weighed 1,011 grammes; right hemisphere, 485 grammes; left, 270; right half of cerebellum, 61; left, 62.

Microscopically, the right cornu ammonis is normal in appearance. In the left, the nerve-cells of the nucleus fasciæ dentatæ are almost entirely wanting. The same is true of the stratum pyramidale with the exception of a small part of its convexity, just before entrance into the fascia dentata. The stratum granulosum is well preserved. The neuroglia of the atrophied portions contains numerous nuclei, but no large spider cells. In specimens stained by Pal's method the deep layer of the alveus and its radiations into the nucleus fasciæ dentatæ are inconspicuous. In the remainder of the cerebrum there seems to be some thickening of the superficial layer of neuroglia in both hemispheres. Otherwise, the right hemisphere seems normal. In the left there is everywhere a diminution of the thickness of the third layer of nerve-cells, and of the number of large pyramidal cells in this layer. The large cells peculiar to the excitable region are well developed. Medullary stains do not bring out any very striking difference in the number or size of the fibres in a given area.

Case 6.—E. P., male, aged 21, died in Danvers Lunatic Hospital Dec. 27, 1895, of diarrhœa. No history of his disease previous to admission was obtained. The right side of the face and right extremities were smaller than the left. There were no contractures, on the contrary, the joints of the right fingers were abnormally loose. There were athetoid movements of right hand. He was almost completely idiotic and talked very imperfectly. He was subject to fits, usually nocturnal, in which he would stand and whirl about, but did not fall.

At the autopsy, after removing the calvarium and dura, there was little difference apparent in the size of the hemispheres, but the left fluctuated when handled. On taking out the brain, the left hemisphere collapsed, a large amount of fluid escaping. The left lateral ventricle was greatly dilated, especially in its posterior part. The entire occipital lobe was very much thinner; over most of the external surface it was only a translucent membrane. The frontal and parietal lobes of this hemisphere were of normal consistency, but the temporal was indurated throughout. Both cornua ammonis were small and indurated. No other localized lesion of the right hemisphere was noticed, but, although symmetrical, it was small, weighing 369 grammes against 187 for the left. The right half of the cerebellum weighed 42 grammes; the left, 58. The entire weight of the brain was 743 grammes. The left optic thalamus and corpus striatum were smaller than the right. In this case, if the left hemisphere had weighed as much as the right, it would have brought the total weight of the brain to only 925 grammes—a distinctly microcephalic weight. In view of this fact, and of the condition of the cornu ammonis, it seems probable that both hemispheres were affected, in this case, by the morbid process.

Microscopically, in both cornua ammonis, there is absence of most of the large nerve-cells of the nucleus fasciæ dentatæ and stratum pyramidale. Stratum granulosum well preserved. Nuclei of neuroglia in atrophoid portions numerous, but spider cells absent. The left gyrus hippocampi, in a portion adjacent to the cornu, is similarly atrophied in its whole thickness. Medullary staining shows the deep layer of the alveus and its radiations into the fascia dentata to be almost absent in the right cornu and greatly thinned in the left.

In the foregoing six cases of infantile hemiplegia, the essential nature of the lesion was evidently the same in all cases, although the reduction in size of the hemisphere took place sometimes more on the outer, at others on the inner surface. It seems to me altogether most probable that the lesion of the cornu ammonis of the affected hemispheres was due to the same cause, whatever that may have been, as that of the other convolutions, rather than that it was an independent change. In the remainder of the hemispheres the appearances varied too greatly for concise description. Where there was complete destruction of the nerve-cells of the cortex, the histological apparances were identical with those of the diseased parts of the cornua.

To be continued.

STUDY OF THE HANDS IN ACROMEGALY BY MEANS OF THE ROENTGEN RAYS. G. P. Marinesco. Comptes Rendues de la Société de Biologie. June, 1896.

The author records four cases which he observed, one apparently of the "giant" type and the others probably of the thick set or "clumsy" type. In the first there was no new osseous formation of the bones at the extremities. The others showed some hypertrophy of the epiphyses, therefore the conclusion was reached that in the "clumsy" type the hypertrophy of the soft parts of the bones is greater than in the "giant" type.

In a case of erythromelalgia which also came under the author's observation, no skeletal changes were to be noted, thus showing that it was not to be classed with acromegaly. JELLIFFE.